HYSTERICAL FEVER.[1]

By MARY PUTNAM JACOBI, M. D.

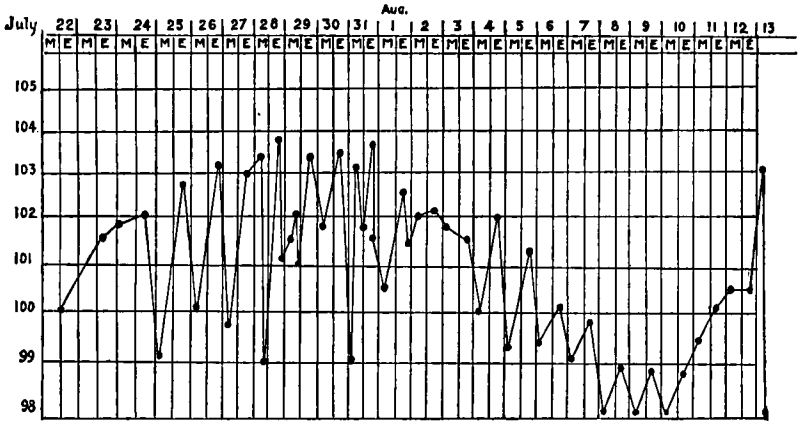
CASE M. M.—The illness to be described began on July 22d last, 1889, when the patient entered the New York Infirmary. But in the preceding year, winter of 1887–1888, she had suffered from a succession of disorders, to which reference must be made on account of their bearing on the illness in question.

In December, 1887, the patient was seized with a pain in the chest, unaccompanied by fever, but which was diagnosed pleurisy by the first physician consulted. His diagnosis had greatly alarmed the patient. As however, my own examination of the chest failed to discover any physical sign of pleurisy, I interpreted the pain as a pleurodynia. After this diagnosis, the pain rapidly subsided; but a paresis of the bladder, which had already showed itself, deepened to a complete paralysis and retention of urine. Catheterism was performed for some time, but the trouble finally yielded to strychnine and local faradization. There appeared severe pain in the left ovarian region, attended with fever. The temperature rose and fell irregularly through the day, occasionally going as high as 103°, more often reaching no higher maximum than 102°. Physical examination of the pelvic organs failed to discover any objective sign of local inflammation, and the disease finally subsided. Before the patient had left her room, however, she was attacked with a severe catarrhal sore throat, attended with abundant diffuse mucous exudation, but not truly diphtheritic. This was in March. After recovery and resumption of ordinary occupations, the patient became subject to intermenstrual metrorrhagia, for which no uterine cause could be ascertained, and which was referred to one of the obscure forms of functional ovarian irritation. During the summer of the

[1] Read at the Neurological Section of the Academy of Medicine, April, 1890.

same year (1888), the patient suffered from a bilateral partial paralysis of the lower extremities, especially affecting the peroneal muscles. She remained able to move her limbs in bed, but was quite unable to stand or walk. She recovered this power, however, when provided with braces which supported the ankles and reaching to the knees. She then went to the seashore, and for two or three months was perfectly well. On returning to the city and becoming involved in much mental worry and anxiety, her physical troubles returned. There were first, attacks of retention of urine and metrorrhagia; then almost entire inability to use the eyes in reading, which a competent oculist explained by simultaneous paresis of several external ocular muscles. He referred this, moreover to an attack of diphtheria which had been experienced five years before; but I think this was improbable, as, until the period which I have just described, the use of the eyes had been attended by no difficulty. It seems to me that the paresis was of an hysterical nature and analogous to that of the peroneal muscles, which had deprived the patient for a time of the power of walking. This opinion was subsequently also expressed by Dr. Putnam, of Boston. The ocular defect persisted through the winter. Twice during this time the patient was seized with an attack of severe pain in the abdomen, which, after lasting twenty-four hours, at once lost its acuity and rapidly subsided, when I had assured her with great positiveness that she did not have peritonitis. Once, after receipt of an agitating letter, she became apparently delirious and unable to speak for twelve hours. This was immediately followed by an intense dysphagia, overcome at last by a combination of moral force and local faradization. In the following summer, 1889, the patient's health was considerably improved. She engaged in some occupation involving considerable fatigue—I think teaching in a public night-school—and at once began to lose ground again. Early in July, during the second day of a menstrual period, she accompanied a friend on an excursion to Bedloe's Island, and climbed the stairs within the statue. The menstrual flow was immediately arrested,

and severe pain appeared in the left ovarian region. On the 22d of July she was admitted to the New York Infirmary, and on the 23d had a temperature of 102°, and the next day of 103°. The fever persisted at about this range till the 13th of August, when, after two days of normal temperature, it rose once more to 103°, and thence fell to a range between 98° and 101.5°, which it maintained till the middle of September. During this time I did not see the patient,



as I was absent from the city. The physicians in charge had only a very slight acquaintance with the previous history of the patient, and she herself gave an imperfect and rather misleading account of her series of illness. On account of the fever, the abdominal pain was explained by some focus of parametritis, but it was noted that the pelvic examination—made, it is true, with reserve on account of the acuity of the accidents—always failed to detect any evidence of inflammatory exudation.

On my return in the middle of September, the patient was in about the same condition as at the beginning of the attack, and quite the same as in the middle of August, after the fever had fallen to a low grade. Upon hearing the history and combining it with that of the many and varied attacks which I had previously and minutely observed, I ventured to express the positive opinion that on this occasion also no really inflammatory process had ever existed,

but that the accidents were nervous, and initiated by an ovarian irritation, the latter due to the arrest of menstruation by an unwonted physical exertion which involved the nerves of the lower extremities, *i. e.*, of the lumbar plexus, which also innervates, to a great extent, the ovary. It seemed probable that the menstrual arrest had left a congestion of the ovarian cortex, or even that minute hemorrhages had occurred there. The patient was put under ether, and a most thorough pelvic examination made, both by myself and by Dr. Cushier, with a completely negative result. Dr. Cushier admitted that the entire absence of any trace of exudation at this time, though some irregular low fever persisted and the abdominal pain was as severe as ever, rendered it altogether improbable that a parametritis had ever occurred.

With the concurrence of Drs. Cushier and Kilham, therefore, I positively assured the patient that she had no pelvic inflammation, that the attack was of the same nature as the others in which I had previously attended her; that she could safely get up from bed as soon as she pleased; and that a few applications of galvanism to the abdomen over the seat of the pain would rapidly dissipate it.

The applications were, in fact, made with the positive electrode over the ovarian region of the abdomen, the negative over the lumbar spine. Each application entirely removed the pain for many hours. But it seems probable that the moral effect of the diagnosis was quite as important, so rapidly did the patient change her attitude and so soon was she able to get out of the bed on which she had been lying for two months. In a week she was walking about; in ten days was entirely free from pain. The temperature remained normal from the day of the examination under ether.

Before the modern researches upon fever as the result of poisonous material circulating in the blood, the conception of a purely "nervous fever" was an entirely familiar one. Indeed the abdominal typhus, which is now recognized as a typical example of infectious disease, was considered, not so very long ago, as a "nervous fever," and

liable to be produced by causes which greatly fatigued or exhausted the nervous system.

The well-known urethral fever was an admitted case of a purely nervous fever of reflex origin. "Febrile movements" of all kinds were easily explained by varying functional irritations of the nervous system, among which were not reckoned irritaments conveyed to nerve centres in the blood nourishing them. Indeed, even the fever of inflammations was referred to the peripheric irritation of the nerves of the inflamed tissues; and not until much later was it suggested that some *materies morbi* was carried from the focus of inflammation to the central nervous system.

To-day, however, the point of view has so radically changed, that it is easy to forget that all the modern explanations of fever simply increase the list of irritaments to which the pyrogenic apparatus of the nervous system is susceptible. Although there be, as there undoubtedly is, increased production of heat during fever, it is established that this would not cause a rise of body temperature unless the elimination of heat were simultaneously deranged absolutely or relatively. But this derangement in the elimination of heat depends upon disorder of the heat-regulating apparatus of medullary and cerebral centres, which thus react to the influence of the chemical poisons generated by inflammation or infection. There is, therefore, no essential contradiction between the new and old views about fever. An exclusively nervous cause is always plausible, because the proximate cause of increased body heat is always to be sought in the nervous system.

Before the thermometer was supposed to enable us to differentiate with precision between inflammatory and non-inflammatory pain, the liability of hysteria to simulate inflammations, and especially those of the abdominal cavity, was one of the well-worn themes of text-books. "Hysterical Peritonitis" is a classical chapter in every dissertation on hysteria, and in every guide to differential diagnosis in abdominal disease. But I think that to-day—and the case I have related shows it—we are liable some-

times to be misled by an habitual, though legitimate, reliance on the thermometer as a means of differentiation. It is easy to decide in the absence of fever that pelvic pain must depend upon some other cause than inflammation; and in the great majority of cases this conclusion is confirmed by the absence of all physical signs of exudation. Yet Dr. Thomas and some other gynecologists declare that an extensive pelvic exudation may be formed, and with considerable rapidity, without the slightest rise of temperature ever being produced. However this may be—and I confess never to have myself seen the statement proved—the two attacks of pseudo-parametritis attended by fever, which were sustained by the highly hysterical patient under discussion this evening, serve to illustrate the converse proposition, namely, that a rise of temperature may occur under circumstances strongly suggestive of pelvic inflammation and yet all positive proof of true inflammation be entirely lacking.

Hysterical fever has lately received much attention from both English and French physicians. In 1883 Pinard wrote a thesis on the pseudo-fever of hysterics, in which he claimed to show that hysterical fever did not really exist:—that is, in the cases described: either no thermometrical observation had been taken, or the thermometer registered a temperature not above 38° C., while often the temperature remained normal. The pseudo-fever consisted, therefore, in an assemblage of symptoms which simulated fever, but were not truly febrile. Among these was conspicuous the acceleration of the pulse, phenomenon essentially analogous to the tachycardia of exophthalmic goitre. The patients often had subjective sensations of heat, also severe headache and coated tongue. This condition was not unfrequently regularly paroxysmal, so as to simulate attacks of malarial fever, but was entirely uncontrolled by quinine.

In a more recent thesis, passed by Henri Fabre in 1888, the existence of a true fever, and even hyperpyrexia of really hysterical origin, is, however, formally reasserted. Cases are related where such fever was accompanied by functional disturbance of various organs, so as to simulate

respectively meningitis, peritonitis, or pneumonia. Intermittent fever and typhoid fever are also said to be simulated. The same assertion is made by an American physician, Bressler, in a communication to the "Medical Record," for 1888. This writer relates no cases in detail, and I do not think that his diagnosis is absolutely proved by his descriptions.

"By hysterical fever," says Dr. Bressler, "I mean a perverted condition of the nervous system, occurring in a neurotic individual, attended by an elevated temperature, which may last from a few hours to several days, and is associated throughout its duration with symptoms of an hysterical character." "This fever," continues the writer, "generally begins with symptoms simulating a mild intermittent—chilliness, loss of appetite, constipation, or occasional diarrhœa; tongue coated, headache, general malaise, rise of temperature, face flushed generally, or in a circumscribed spot on the cheeks, eyes clear and brilliant, mind bright, comprehension quickened. There is general muscular and cutaneous hyperæsthesia. The special senses are more acute; there is no true delirium. The stomach is excessively irritable, and vomiting very persistent. The abdomen is extremely sensitive to pressure, and peritonitis may be simulated, but may be excluded by the fluctuating character of the pains, the absence of tympanitis, and the development of ovarian pain under pressure. The temperature varies from 101° to 105° F., and the maximum is reached early in the attack."

In the "Transactions of the London Clinical Society," Dr. Hale White relates the following case: A girl of eighteen was admitted to the ward, on the 10th of August, for a febrile attack, which lasted four days, and then subsided. On September 8th she was suddenly taken ill with a severe pain in the left side, and was readmitted to the hospital the next day. The patient could hardly walk, and was somewhat incoherent in speech. Within the course of twelve hours the pain was located in four different places—the left iliac region, the epigastrium, the lumbar region, the splenic region. The attention of the patient was easily diverted by

conversation, and she then permitted considerable pressure over the seat of the pain. The temperature was at first 103°; on September 10th, after a chill, rose to 105°, to fall in the evening to 99°. On the 11th, at 6 A. M., the temperature was 98.6°, at 6 P. M. 104°; September 12th the temperature did not rise till evening, when it was 102° at 6 and 98.8° at 10.

In the analysis of the case Dr. White excluded all other causes of either the pain or the fever except hysteria. But it is noticeable that the patient vomited on two successive days, and during the previous brief illness in August there had also been symptoms of a gastro-duodenal catarrh. It seems to me that such an organic condition really existed, and was the immediate cause of the neurotic condition upon which the wandering pains, and markedly irregular fever, directly depended.

Dr. White remarks that, although several cases of hysterical pyrexia have lately been recorded, much skepticism has been expressed in regard to it. Among these recorded cases is one by Clemrow, in the "Medical Press and Circular," of 1887. A laundrymaid, of twenty-three, was admitted to the Edinburgh Royal Infirmary, October 22d, with dizziness, pain in the left side, and a purpuric rash over the lower extremities. On the 29th of November the patient had a severe fright, and her temperature rose to 107.8°. After this the records of temperature are so extraordinary as to suggest fraud, were it not that there was no way in which a fraud could have been effective. At midnight of the same day three successive records, taken at short intervals, read 111°, 108°, 98°. On November 30th the temperature in the right axilla was 108°; the left, at the same time, 99.4°. At midnight the temperature was 98° on the right side and 108° on the left. Similar local maxima, varying from hour to hour, were observed on the 1st, 2d, and 3d of December; after which the records are not given. On November 30th the patient had several spasms simulating tetanus, probably hysterical opisthotonos. On December 1st, together with headache and nausea, there was a peculiar rhythmical movement of the eyelids, alternate

elevation and depression. There was left internal strabismus, and sluggish reaction to the light of the right pupil. Throbbing pain at the vertex increased by pressure. On December 3d there were frequent spasms, with muffled heart-sounds; pulse at the wrist imperceptible. On December 4th the patient became delirious, and continued so until the 13th. The plantar and patellar reflexes were both absent; there was cutaneous anæsthesia, incontinence of urine and fæces. After the 13th these symptoms disappeared, and the patient began slowly to improve. But she was not fully recovered until April.

Clemrow considered the hyperpyrexias to have been local, and not extending throughout the body.

In the "Lancet," for 1879, Donkin related the case of a girl of nineteen, who, during convalescence from a mild typhoid fever, had, at frequent intervals, temperatures of 108° or 110°. These were of short duration, and unaccompanied by other symptoms than a sensation of heat.

In another case, observed by the same writer, from the 20th of May to the 20th of June the temperature every morning and evening varied between 101.8° and 106.8°.

Donkin quotes similar cases from Creig Smith, Cliffe, and Meade. The last, like Donkin's own case, was also a girl convalescent from typhoid, whose temperature for a month kept incessantly varying from 103° to 109°, sometimes in fifteen minutes would run up to 111°. In these English cases the temperature was always taken in the axilla.

In the "Gazette Hebdomadaire," for 1886, Debove describes a patient who, every day for a month, and without other symptom, presented morning and evening a temperature of 39.5° C. This was in November. In December the temperature rose to 40°, on the 17th of January was 41.4°, and on the 24th reached a final maximum of 41.4°. After this it slowly fell, and became normal on the 30th. During this period of three months the morning and evening temperatures were almost always alike: occasionally one or the other was higher by one-tenth or two-tenths of a degree. This prolonged hyperpyrexia resulted in no emaciation or loss of strength.

In 1886 Barié described a case (also in "Gazette Hebdomadaire"), a severely hysterical young woman, servant at Bicêtre. She was subject to frequent convulsive attacks, transient paralyses, profound disorders of sensibility. One morning, after a violent convulsion, she became completely hemiplegic, on theleft side, except the face, as regarded both mobility and sensibility. After this she had thirty convulsive attacks in the course of twelve days. Sometimes for two or three days together she would remain in a state of complete mutism, without eating and also without urinating. All remedial measures failed, and the physician contented himself with simple observation. One morning, after a violent convulsive attack, the temperature in the axilla was found to be 39° C. From this time, for twenty days, there was permanent fever, as measured both in the axilla and rectum. Evening temperature was usually higher than morning by some tenths of a degree, but on five days the morning temperature was the highest. There was no functional disturbance, and the tongue remained moist. The fever was highest on the days of the attacks, but persisted on the other days also. On the twentieth day sudden defervescence occurred, the patient remaining otherwise the same, neither better nor worse.

In the "Periscope" of the JOURNAL OF NERVOUS AND MENTAL DISEASE, for February, 1890, is described a case of hysterical pseudo-phthisis where, during three days, the temperature varied from 103° to 104° F.; on the fourth day it rose to 113°, and the patient became slightly delirious. In an hour the temperature fell to 108°; in the evening was 106.3°. On the next day it again rose to 113°, but fell in an hour to 99.5°. During the next few days the temperature varied from 101.3° to 103.1°, and then became normal. The symptoms had begun with an attack of hæmoptysis, which was followed by severe dyspnœa, cyanosis, and apparently threatened asphyxia several times during the night. During the next two months the same group of symptoms was repeated several times with complete absence of physical signs of phthisis. There was retention of urine.

The most interesting cases quoted in the thesis of Henri

Fabre are two, of simulated meningitis, one of apparently severe pulmonary disease. The first of these, a young woman of twenty-four, who had previously suffered from chorea and nervous aphonia, was admitted to the hospital with a temperature of 39.5° C. Her face was swollen and congested, eyes closed on account of an intense photophobia. The head was retracted completely, cephalalgia violent, insomnia and cries, abdomen retracted, constipation absolute, meningitic streak easily developed, severe generalized hyperæsthesia, knee-jerk little modified, no morbid condition discoverable in lungs, heart, or kidneys. During ten days the patient remained in about the same condition: prostrated, eyebrows contracted, pupils contracted but equal, five or six times bilious vomiting without effort (having all the appearance of cerebral vomiting). A diagnosis was made of tubercular meningitis, and (but with little hope of doing any good) leeches were applied behind the ears and calomel administered. On the tenth day the patient was found sleeping naturally, and, on being aroused, ceased to complain of the pain in her head. The temperature had fallen to 38° C. In a few days more the patient was fully convalescent, but on first getting up was affected by a transient paraplegia.

The history of the second case closely resembled the first.

I have myself seen a similar case in the service of Cornil at La Charité, and, curiously enough, the same patient returned, a year later, with the same group of symptoms, and, her personality being recognized, the diagnosis was the second time at once correctly made.

The case of febrile hysteric dyspnœa related by Fabre is as follows: The patient was a woman of twenty-six; admitted to the hospital with an evening temperature of 39° C. and a dyspnœa of five or six days' duration. There were thirty-five to forty respirations a minute, but unaccompanied by trace of cyanosis. The most careful auscultation failed to discover any lesion of either lungs or heart, and the absence of albuminuria was held to exclude a uræmic origin to the dyspnœa. The fever continued for twenty

days, being extremely irregular, with occasional intermissions of normal temperature, followed by a rise to 39° or 40° or over. On the twentieth day occurred an abrupt defervescence, and at the same time the dyspnœa ceased.

The recognition of hysterical fever as a distinct clinical affection has been much facilitated by recent researches on the relations of the cerebro-spinal nerve-centres to the temperatures (general or local) of the body. As every one knows, these researches were initiated by the famous observation of Sir Benjamin Brodie, on a rise of temperature in a few hours to 111° F., in a patient who had sustained a fracture of the spine, with traumatic section of the cord. This observation was published in the "Medico-Chirurgical Transactions" in 1837.

The researches of Tscheschin, in 1866, are equally famous and well known. In some respects they seem in contradiction with Brodie's clinical observation: for when, in animals, this experimenter cut the spinal cord below the medulla, the temperature of the body fell; but if the section were made between the medulla and the pons, the temperature rose excessively.

The more exact experiments of Horatio Wood, in his beautiful researches on fever, published in 1880, demonstrated that when the spinal cord was cut anywhere between the level of the third and second cervical vertebra there was at first an enormous increase of heat-dissipation, correlative with the general vaso-motor paralysis; that in forty-eight hours this was followed by a diminution in the dissipation of heat, but also a diminution in heat-production, so that, as had been before observed, the net result was a fall of body temperature. Wood also observed the rise of temperature consecutive to section of the cord between the medulla and pons. He accepts the inference drawn from the facts by Tscheschin, that there exists in the medulla some nerve centre or centres whose influence tends to stimulate the production of heat in the thermo-genetic tissues, namely, the muscles; that this influence is habitually restrained by that of moderating centres in the pons or above it, and that the rise of temperature observed in

the last experiment is due to the withdrawal of this moderating influence from the real heat centres. More recent experiments have extended the field of experiment and inquiry. Eulenburg and Landois showed that excitation of one cerebral hemisphere is followed by a local rise of temperature in the limbs of the opposite side. These experimenters made no observations on the general temperature. In 1884, Charles Richet (Compt. Rend. Societé Biol., 22 Mars, 1884) pricked one cerebral hemisphere of a rabbit with a steel pen which perforated the cranium, and and found in the course of two hours that the rectal temperature rose from 39.5° C. to 40.4°. The next day, when the temperature had fallen to 39.2°, a nerve pricking caused a rise to 42.8°. The animal died in the night, presumably of the hyperpyrexia, as no brain lesions were discovered to explain the death. It was found that the pin had penetrated to a spot situated three or four millimetres in front of the corpus striatum.

A little later, Schreiber[2] found that a rise of temperature occurred after lesion of any part of the pons, of the cerebral peduncles, cerebrum or cerebellum, provided the animal operated on were protected from the radiation of heat by wrapping in cotton wool. In 1885, Aronsohn and Sachs in Germany, and Dr. Isaac Ott in America, began almost simultaneously, but quite independently of each other, to search for heat-regulating centres in the brain. The German observers[3] trepanned rabbits at the juncture of the sagittal and coronal sutures, and entered the brain with a needle, three millimetres broad, at a point about one millimetre outside the longitudinal sinus. A carbolized dressing was immediately applied, and the well-being of the animals seemed to remain undisturbed.

When the operation was performed on the cerebrum anterior to the Rolandic convolutions, no effect on the temperature was observed. But the punctures which passed to the base of the brain, from the point of junction of the coronal and sagittal sutures, were always followed by an

[2] Pfluger's Archiv., viii., S. 576.

[3] Pfluger's Archiv., 1885.

enormous rise of temperature. If the puncture only penetrated the cortex cerebri, no effect on temperature was produced. Electrical irritation of the susceptible region, *i. e.*, the tissue just in front or on the outer side of the corpus striatum, also caused a rise of temperature. An increased excretion of nitrogen was observed during this artificial fever, so an increased heat-production was inferred, but no calometrical observations were made.

These difficult observations were, however, made by Ott,[1] and add greatly to the value of his experiments on the brain.

Ott established four localities at the base of the brain whose puncture, and consequent irritation, was followed by a rise of body temperature. These were, at a point just within the anterior part of the corpus striatum; a second point between the corpus striatum and the thalamus; a third at the anterior part of the thalamus; and a fourth at the point of decussation of motor fibres at the nib of the calamus in the medulla. In the fever consecutive to irritation of these centres, there is at first an increase of both heat-production and heat-dissipation, but both soon fall below normal, though fever continues. In addition to these centres, however, Ott discovered two others on the cortex; one at the point of juncture of the supra sylvian and post sylvian fissure; the other in the neighborhood of the cruciate sulcus, *i. e.*, over the Rolandic convolutions.

When either of these cortical centres were irritated, temperature was depressed. If, on the other hand, they were removed by slicing and subsequent washing with carbolized water, the temperature rose.

From the total result of his experiments, Ott infers that the basal centres, like those of the spinal cord, habitually stimulate the production of heat; are thermogenetic centres. But those of the cortex, the sylvian and cruciate, habitually restrain the activity of these lower centres, and may therefore be called thermotaxic.

Under certain circumstances the striate and extra striate centres may also be thermotaxic, and moderate the

[1] Journal Mental Disease, 1888.

spinal centres below them. They have, therefore, a mixed character or function.

Girard[5] confirmed the results of Ott's experiments on the corpus striatum, and also observed a rise of temperature to follow punctures at various localities in the posterior part of the brain, but none when these were made anteriorly. The fever was attended by increased elimination of nitrogen in the urine, and was controlled by antipyrine. Rise of temperature was also induced by faradising the striated bodies for half an hour with needles insulated to their tips.

Horatio Wood, also, in thirteen out of fourteen experiments, found that localized destructions of tissue just behind the crucial sulcus, thus compromising Hitzig's region, were followed by a rise of temperature and decided increased of heat-production.

A curious confirmation of the foregoing observations is offered by Zawadowski,[6] who found that antipyrine ceases to reduce temperature if administered after section of the spinal cord at the atlas, an operation which removes the inhibitory influence of the brain from the thermogenetic centres of the cord.

The interest of the foregoing observations is very great in their bearing on the general theory of fever. In accordance with them, all fever can finally be ascribed to derangement of the central nervous apparatus, which controls the generation of heat in the muscles, the latter being the ultimate thermogenetic apparatus. Hence, the striking fact, that the cerebral centres so far established as regulating the production of heat, are chiefly situated on the motor tracts, namely the Rolandic convolutions, the striate centres, and the medulla.

In zymotic fever the thermogenetic centres would be irritated by the poison circulating in the blood; in traumatic, perhaps also in inflammatory fever, the same result is produced by irritation of peripheric nerves; in hysteria there would be paralysis of the cortical thermotaxic inhibitory centres rather than excitation of the basal thermo-

[5] Archives to Physiol., 1886 and 1888.

[6] Centralblatt f. medicim-wissen, 1888.

genetic centres.[7] Reflex fevers, like urethral and worm fever, might be supposed to imply, on the other hand direct irritation of the thermogenetic centres.

This paralysis would then enter into the entire series of hysterical phenomena, which depend upon loss of cortical control over lower centres. It becomes analogous to the loss of cortical control over subcortical vaso-motor centres, upon which Meynert has so strongly insisted, and nevertheless it is not to be resolved into a vaso-motor phenomenon. For it has been shown, especially in some experiments of Wood's, that the vaso-motor medullary centres are not affected in these artificial fevers, and respond as usual to an irritation of the sciatic nerve.

A danger attends the recognition of any group of clinical symptoms as hysterical. It is the danger of ascribing to hysteria, symptoms which are really caused by organic disease. This is even more serious than the opposite error of interpreting as the result of organic disease, symptoms really due to hysteria. The diagnosis is, therefore, always important, and often delicate and difficult. It would be impracticable in this place to analyze the elements of diagnosis in regard to each case which might be simulated. But this may always be remembered: Exclusion of the grave organic lesion which may be simulated, does not necessarily exclude the origin of the disorder in some lesser lesion, which may even entirely disappear, while the storm which has been aroused continues. The type of such a sequence is offered by the prolonged hysterical neuralgias which may originate in a slight sprain (traumatic hysteria).

In the case which forms the basis of this paper, I think it is not at all improbable that the last series of accidents originated in a slight hemorrhage into the cortex of the ovary, occurring at the time of the arrested menstruation. A permanent ovarian irritation or irritability existed, manifested by the persistent recurrence of menorrhagias, in the absence of all uterine disease. It seems as if this would be sufficient to explain the entire series of phenomena, itself being an expression of a grave hysterical diathesis.

[7] W. Hall White, loc. cit.